

Carbohydrate Content in the GDM Diet: Two Views

View 2: Low-Carbohydrate Diets Should Remain the Initial Therapy for Gestational Diabetes

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■ **IN BRIEF** The appropriate dietary intervention for gestational diabetes mellitus (GDM) is not clear. Traditionally, a low-carbohydrate diet has been prescribed. Recently, there has been a movement to prescribe a diet higher in nutrient-dense carbohydrate as the initial treatment for GDM. At this time, there is insufficient outcome data to support this type of diet.

Gestational diabetes mellitus (GDM) is a common disorder of pregnancy. Approximately 6–7% of all pregnancies are affected by diabetes, of which 90% are considered GDM (1). The number of women affected by GDM is expected to increase as more obese and morbidly obese women achieve pregnancy. In addition, if the criteria proposed by the International Association of Diabetes and Pregnancy Study Groups are adopted by the American College of Obstetrics and Gynecology (ACOG), there will be a surge in the number of women diagnosed with this disorder in the United States (1,2).

The risks of GDM are well defined. It is associated with both antenatal and neonatal risks for the offspring, as well as possible lifelong health concerns. The most common obstetrical risks include hypertensive disorders of pregnancy, dystocia, cesarean delivery, and pelvic trauma. The fetal and neonatal risks include macrosomia, birth trauma, hypoglycemia, metabolic disorders, hyperbilirubinemia from polycythemia, and intracardiac septal hypertrophy. Long-term maternal concerns include increased risks of type 2 diabetes and cardiovascular disease. The risks to offspring of mothers with diabetes include

childhood and adult obesity, type 2 diabetes, and hypercholesterolemia. All of these risks appear to be directly or indirectly related to glucose metabolism during pregnancy.

Treatment goals of GDM include the avoidance of fetal hyperinsulinemia, which leads to macrosomia, birth trauma (neonatal and maternal), and cesarean delivery. All of these complications lead to poor neonatal outcomes and maternal complications. Although the goal is fairly clear—prevent fetal hyperinsulinemia and macrosomia—the way to achieve this goal is not. Pedersen (3), in 1952, theorized that maternal hyperglycemia leads to fetal hyperglycemia and fetal macrosomia. Combs et al. (4), in 1992, compared pre- and postprandial blood glucose levels to fetal macrosomia in patients with pregestational diabetes. This study showed a positive relationship between fetal macrosomia and maternal glucose control, and specifically postprandial glucose control. The 2008 Hyperglycemia and Adverse Pregnancy Outcomes (HAPO) study (5) was designed to evaluate the effect of maternal hyperglycemia. This study demonstrated the association between elevated blood glucose levels with neonatal complications. Maternal hyperglyce-

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mia was associated with an increased risk of macrosomia, primary cesarean section, neonatal hypoglycemia, and hyperinsulinemia. Elevated blood levels of fasting, 1-hour, and 2-hour post-load plasma glucose obtained through oral glucose tolerance testing were linked to birth weights and cord-blood serum C-peptide levels >90th percentile. Less significant associations were noted between glucose levels and primary cesarean delivery and clinical neonatal hypoglycemia. Increased plasma glucose levels were also associated with increased risks of premature delivery, shoulder dystocia or birth injury, intensive neonatal care, hyperbilirubinemia, and pre-eclampsia. Crowther et al., in the Australian Carbohydrate Intolerance Study in Pregnant Women (6), sought to determine whether treatment of GDM could lower the risks of shoulder dystocia and neonatal macrosomia. This study documented significantly lower complications with decreases in maternal hyperglycemia. Subsequently, Landon et al. (7,8) showed that treating mild GDM with diet, self-monitoring of blood glucose, and insulin therapy to reduce hyperglycemia and prevent postprandial hyperglycemia significantly decreased the risk of fetal overgrowth, shoulder dystocia, cesarean delivery, and hypertensive disorders.

Diet therapy is generally the first-line treatment for women with GDM. The traditional diet is based on body weight using a formula of kcal/kg. Once the overall energy needs are determined, the general composition is 40% carbohydrate, 15–20% protein, and 40–45% fat. More recently, this prescription has come into question. In 2011, the American Diabetes Association (ADA) removed the recommendation for a specific diet or a specific carbohydrate intake from its practice guidelines (9). Its most recent statement recommends a diet that promotes maternal and fetal well-being in the absence of ketones, but allows for adequate caloric consumption for adequate maternal weight

gain. ACOG, however, continues to recommend a low-carbohydrate diet, with carbohydrate comprising 35–40% of diet intake. The rationale for this is that increased levels of carbohydrate in the diet increases maternal weight gain and maternal glucose levels, leading to fetal macrosomia (1). This is where the controversy lies. Should practitioners continue to recommend a low-carbohydrate diet in the initial treatment of GDM?

Jovanovic-Peterson and Peterson (10), in 1990, examined the relationship between carbohydrate intake and 1-hour postprandial blood glucose. There was a significant relationship between percentage of carbohydrate and postprandial blood glucose level. Lower-carbohydrate diets yielded lower postprandial glucose levels. Amniotic fluid levels were also measured, and elevated levels of amniotic fluid insulin also were directly associated with carbohydrate intake. Based on their results, these investigators recommended that diet for GDM focus on lowering carbohydrate intake.

Major et al. (11), in 1998, compared diets consisting of <42% carbohydrate and >45% carbohydrate to manage GDM. Although this was a nonrandomized study, it demonstrated a decreased risk of postprandial hyperglycemia, lower risk of requiring insulin to control blood glucose, and lower risk of fetal macrosomia.

In contrast to earlier studies, Cypriak et al. (12), in 2007, compared a lower-carbohydrate diet to a higher-carbohydrate diet and found that, after 2 weeks, there was no significant difference in blood glucose control between the two groups. However, because of this study's short time period, no neonatal or maternal outcomes could be reported. Viana et al. (13) performed a 2014 meta-analysis looking at the effects of dietary intervention on neonatal outcomes and reported that low-carbohydrate diets had no effect on

neonatal outcome; however, based on the paucity of available controlled studies, the data reported were limited. In 2013, Moreno-Castilla et al. (14) compared a low-carbohydrate to a higher-carbohydrate diet to determine the effect on the need to treat GDM with insulin. The primary outcome of this study did not include neonatal complications. A 2009 systematic review of randomized studies (15) addressing dietary treatment of GDM concluded that there was insufficient evidence to recommend a specific dietary approach. The authors reported that there were not enough randomized, controlled studies to support either type of diet.

Hernandez et al. (16) reported the results of a randomized crossover study in which investigators increased the amount of carbohydrate and decreased the amount of fat in patients' diets. They used a higher percentage of more nutrient-dense carbohydrate using the CHOICE (Choosing Healthy Options in Carbohydrate Energy) diet (60% carbohydrate, 45% fat, and 15% protein) with continuous glucose monitoring to determine mean blood glucose. This study was controlled, and carbohydrate intake was closely monitored. The results showed higher 1- and 2-hour postprandial breakfast values, 5-hour postprandial glucose levels, and overall daytime glucose values. Insulin and C-peptide levels on the CHOICE diet were higher than on the low-carbohydrate diet. However, these authors argued that the area under the curve glucose was still within the target range in the group with the higher-carbohydrate diet. They noted that free fatty acids were lower in the higher-carbohydrate diet and suggested that the free fatty acid levels may be an important component of the diet to treat GDM and prevent neonatal adiposity. However, obstetrical and neonatal outcomes were not addressed; this study specifically looked at glucose metabolism and not neonatal or maternal obstetrical outcomes.

Trout et al. (17), in an article published in this issue of *Diabetes Spectrum* (p. 71), compared a low-carbohydrate diet (40% carbohydrate, 40% fat, and 15% protein) to a high-carbohydrate diet. However, the primary outcome of lower 2-hour postprandial blood glucose was not achieved. There was a trend toward lower fasting blood glucose and 2-hour postprandial values when results from the two sites for this study were compared. Although there was no significant difference in the neonatal complications of macrosomia and shoulder dystocia, there was a trend toward neonatal hypoglycemia in the nonintervention cohort following the higher-carbohydrate diet.

Interestingly, there was significantly lower 2-hour postprandial blood glucose in one of the two sites. There was also a significant difference in the rate of neonatal complications when the two sites were compared. Site A (a teaching hospital) is located in a food desert, whereas Site B (a community hospital) had a low poverty rate. When the two sites were compared, carbohydrate intake was similar, but Site A had lower total protein and fat intake than Site B. This study suggests that prescribing a specific carbohydrate diet does not address other factors that affect glucose response. Not only should a low-carbohydrate diet be prescribed, but fat and protein intake also should be addressed to achieve the ADA goal of a diet that promotes maternal and fetal well-being.

Despite advocates for higher-carbohydrate diets suggesting that there is no difference in postprandial blood glucose levels between low- and higher-carbohydrate diets, there is insufficient evidence to support a higher-carbohydrate diet in GDM.

There is also little evidence that a higher-carbohydrate diet prevents the neonatal complications linked to GDM. Although Hernandez et al. (16) do recommend a higher-carbohydrate diet with nutrient-dense carbohydrate, they report that blood glucose levels are higher with such a diet. Higher maternal blood glucose levels lead to fetal hyperinsulinemia, which then leads to maternal and neonatal complications. The main goal of GDM treatment is the achievement of lower postprandial blood glucose levels, with a resultant decrease in the risk of fetal hyperinsulinemia. Until studies can demonstrate that higher-carbohydrate diets do not cause an increase in fetal insulin secretion, low-carbohydrate diets should continue to be recommended. The best way to achieve lower postprandial blood glucose levels and prevent fetal hyperinsulinemia is to prescribe a low-carbohydrate diet.

Duality of Interest

No potential conflicts of interest relevant to this article were reported.

References

1. American College of Obstetricians and Gynecologists. Gestational diabetes mellitus (Practice Bulletin No. 137). *Obstet Gynecol* 2013;122:406–416
2. Jovanovic L. American Diabetes Association's Fourth International Workshop-Conference on Gestational Diabetes Mellitus: Summary and discussion: therapeutic interventions. *Diabetes Care* 1998;21 (Suppl. 2):B131–B137
3. Pedersen J. Course of diabetes during pregnancy. *Acta Endocrinol* 1952;9:342–364
4. Combs CA, Gunderson E, Kitzmiller JL, Gavin LA, Main EK. Relationship of fetal macrosomia to maternal postprandial glucose control during pregnancy. *Diabetes Care* 1992;15:1251–1257
5. Metzger BE, Lowe LP, Dyer AR, et al. Hyperglycemia and adverse pregnancy outcomes. *N Engl J Med* 2008;358:1991–2002

6. Crowther CA, Hiller JE, Moss JR, et al. Effect of treatment of gestational diabetes mellitus on pregnancy outcomes. *N Engl J Med* 2005;352:2477–2486
7. Landon MB, Mele L, Spong CY, et al. The relationship between maternal glycemia and perinatal outcome. *Obstet Gynecol* 2011;117:218–224
8. Landon MB, Spong CY, Thom E, et al. A multicenter, randomized trial of treatment for mild gestational diabetes. *N Engl J Med* 2009;361:1339–1348
9. American Diabetes Association. Standards of Medical Care in Diabetes—2011. *Diabetes Care* 2011;34(Suppl. 1):S11–S61
10. Jovanovic-Peterson L, Peterson CM. Dietary manipulation as a primary-treatment strategy for pregnancies complicated by diabetes. *J Am Coll Nutr* 1990;9:320–325
11. Major CA, Henry MJ, de Veciana M, Morgan MA. The effects of carbohydrate restriction in patients with diet-controlled gestational diabetes. *Obstet Gynecol* 1998;91:600–604
12. Cypryk K, Kaminska P, Kosinski M, Pertynska-Marczewska M, Lewinski A. A comparison of the effectiveness, tolerability and safety of high and low carbohydrate diets in women with gestational diabetes. *Endokrynol Pol* 2007;58:314–319
13. Viana LV, Gross JL, Azevedo MJ. Dietary intervention in patients with gestational diabetes mellitus: a systematic review and meta-analysis of randomized clinical trials on maternal and newborn outcomes. *Diabetes Care* 2014;37:3345–3355
14. Moreno-Castilla C, Hernandez M, Bergua M, et al. Low-carbohydrate diet for the treatment of gestational diabetes mellitus: a randomized controlled trial. *Diabetes Care* 2013;36:2233–2238
15. Alwan N, Tuffnell DJ, West J. Treatments for gestational diabetes. *Cochrane Database Syst Rev* 2009;3:CD003395
16. Hernandez TL, Van Pelt RE, Anderson MA, et al. A higher-complex carbohydrate diet in gestational diabetes mellitus achieves glucose targets and lowers postprandial lipids: a randomized crossover study. *Diabetes Care* 2014;37:1254–1262
17. Trout KK, Homko CJ, Welzel-Effinger L, et al. Macronutrient composition or social determinants? Impact on infant outcomes with gestational diabetes. *Diabetes Spectrum* 2016;29:71–78